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THE INFLUENCE OF CARDIORESPIRATORY FITNESS ON THE
DECREMENT IN MAXIMAL AE..(U) ARMY RESEARCH INST OF
ENVIRONMENTAL MEDICINE NATICK MA A J YOUNG ET AL.

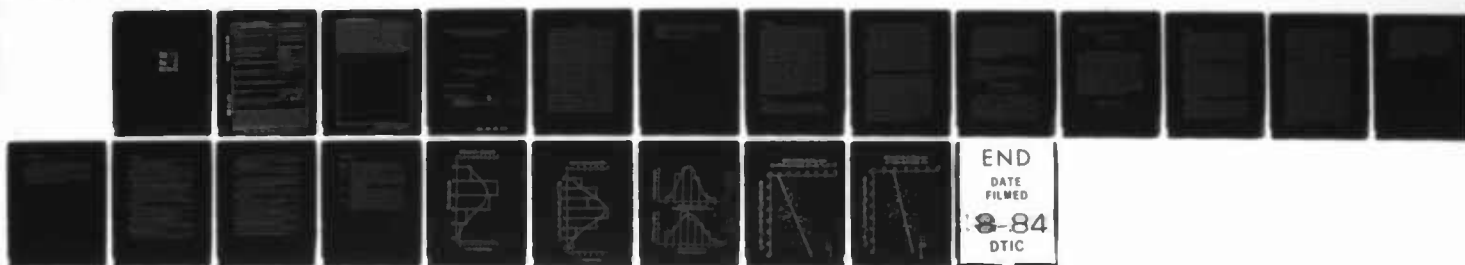
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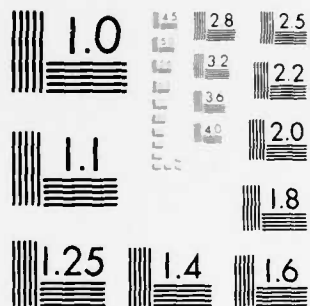
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REPORT DOCUMENTATION PAGE		READ INSTRUCTIONS BEFORE COMPLETING FORM
1. REPORT NUMBER M25/84	2. GOVT ACCESSION NO.	3. RECIPIENT'S CATALOG NUMBER
4. TITLE (and Subtitle) The Influence of Cardiorespiratory Fitness on the Decrement in Maximal Aerobic Power at High Altitude		5. TYPE OF REPORT & PERIOD COVERED
7. AUTHOR(s) A.J. Young, A. Cymerman, R.L. Burse		6. PERFORMING ORG. REPORT NUMBER
9. PERFORMING ORGANIZATION NAME AND ADDRESS USA Research Institute of Environmental Medicine, Natick, MA 01760		8. CONTRACT OR GRANT NUMBER(s)
11. CONTROLLING OFFICE NAME AND ADDRESS Same as 9 above		10. PROGRAM ELEMENT, PROJECT, TASK AREA & WORK UNIT NUMBERS 3E162777A879 44683304126
14. MONITORING AGENCY NAME & ADDRESS (if different from Controlling Office)		12. REPORT DATE 11 Jun 84
		13. NUMBER OF PAGES 14
		15. SECURITY CLASS. (of this report) Unclassified
		15a. DECLASSIFICATION/DOWNGRADING SCHEDULE
16. DISTRIBUTION STATEMENT (of this Report) Approved for public release; distribution is unlimited.		
17. DISTRIBUTION STATEMENT (of the abstract entered in Block 20, if different from Report)		
18. SUPPLEMENTARY NOTES		
19. KEY WORDS (Continue on reverse side if necessary and identify by block number) hypoxia, $\dot{V}O_2$ max, physical fitness		
20. ABSTRACT (Continue on reverse side if necessary and identify by block number) There are conflicting reports in the literature which imply that the decrement in maximal aerobic power experienced by a sea-level (SL) resident sojourning at high altitude (HA) is either smaller or larger for the more aerobically "fit" person. In the present study, data collected during several investigations conducted at an altitude of 4300 m were analyzed to determine if the level of aerobic fitness influenced the decrement in maximal oxygen uptake ($\dot{V}O_2$ max) at HA. The $\dot{V}O_2$ max of 51 male SL residents was measured at an altitude of 50 m and again at 4300 m. The subjects ages, heights, and weights (mean \pm SF) were 22 ± 1 yr \pm 7 cm and 78 ± 2 kg.		

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respectively. Aerobic fitness was expressed as $\dot{V}O_2$ max relative to body mass ($\text{ml} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$). The decrement in $\dot{V}O_2$ max at HA was expressed in terms of both the absolute difference ($\text{ml} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$) between $\dot{V}O_2$ max at SL and HA, and the % of $\dot{V}O_2$ max at SL which was lost at HA. The subjects were normally distributed with respect to their aerobic fitness, ranging from 36 to 60 $\text{ml} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$ (mean \pm SE = 48 ± 1). Likewise, the decrement in $\dot{V}O_2$ max at HA (both computations) was normally distributed from 3 $\text{ml} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$ (9% $\dot{V}O_2$ max at SL) to 29 $\text{ml} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$ (54% $\dot{V}O_2$ max at SL), and averaged $13 \pm 1 \text{ ml} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$ ($27 \pm 1\%$ $\dot{V}O_2$ max at SL). The linear correlation coefficient between aerobic fitness and the magnitude of the decrement in $\dot{V}O_2$ max at HA expressed in absolute terms was $r = 0.56$, or expressed as % $\dot{V}O_2$ max at SL was $r = 0.30$; both were statistically significant ($p < 0.05$). Therefore, only 31 and 9%, respectively, of the variability in the decrement at HA could be attributed to the $\dot{V}O_2$ max at SL. Thus, the level of aerobic fitness per se accounts for a relatively small amount of the variability between individuals in the decrement in maximal aerobic power which occurs at high altitude.

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THE INFLUENCE OF CARDIORESPIRATORY FITNESS ON THE DECREMENT
IN MAXIMAL AEROBIC POWER AT HIGH ALTITUDE

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Running Head: $\dot{V}O_2$ max at High Altitude

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SUMMARY

There are conflicting reports in the literature which imply that the decrement in maximal aerobic power experienced by a sea-level (SL) resident sojourning at high altitude (HA) is either smaller or larger for the more aerobically "fit" person. In the present study, data collected during several investigations conducted at an altitude of 4300 m were analyzed to determine if the level of aerobic fitness influenced the decrement in maximal oxygen uptake ($\dot{V}O_2$ max) at HA. The $\dot{V}O_2$ max of 51 male SL residents was measured at an altitude of 50 m and again at 4300 m. The subjects' ages, heights, and weights (mean \pm SE) were 22 ± 1 yr, 177 ± 7 cm and 78 ± 2 kg, respectively. Aerobic fitness was expressed as $\dot{V}O_2$ max relative to body mass ($\text{ml} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$). The decrement in $\dot{V}O_2$ max at HA was expressed in terms of both the absolute difference ($\text{ml} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$) between $\dot{V}O_2$ max at SL and HA, and the % of $\dot{V}O_2$ max at SL which was lost at HA. The subjects were normally distributed with respect to their aerobic fitness, ranging from 36 to 60 $\text{ml} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$ (mean \pm SE = 48 ± 1). Likewise, the decrement in $\dot{V}O_2$ max at HA (both computations) was normally distributed from 3 $\text{ml} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$ (9% $\dot{V}O_2$ max at SL) to 29 $\text{ml} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$ (54% $\dot{V}O_2$ max at SL), and averaged 13 ± 1 $\text{ml} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$ ($27 \pm 1\%$ $\dot{V}O_2$ max at SL). The linear correlation coefficient between aerobic fitness and the magnitude of the decrement in $\dot{V}O_2$ max at HA expressed in absolute terms was $r = 0.56$, or expressed as % $\dot{V}O_2$ max at SL was $r = 0.30$; both were statistically significant ($p < 0.05$). Therefore, only 31 and 9%, respectively, of the variability in the decrement at HA could be attributed to the $\dot{V}O_2$ max at SL. Thus, the level of aerobic fitness

per se accounts for a relatively small amount of the variability between individuals in the decrement in maximal aerobic power which occurs at high altitude.

Key Words: Hypoxia, $\dot{V}O_{2\max}$, Physical Fitness

INTRODUCTION

The maximal oxygen uptake ($\dot{V}O_2$ max) of sea level residents is reduced by exposure to hypoxia. The amount of reduction is proportional to the reduction in the partial pressure of oxygen in the inspired air. The relationship between the reduction in $\dot{V}O_2$ max and altitude has been empirically determined to be on the order of 10% (of sea level $\dot{V}O_2$ max) for every 1000 m ascended beyond an altitude of 1500m above sea level, with little or no apparent decrement in $\dot{V}O_2$ max between sea level and 1000 m (Buskirk 1969; Hartley 1971). There is, however, considerable individual variability in the magnitude of this decrement at high altitude. This variability is particularly significant at moderate altitudes (1000-2000 m) where the decrement is small (Squires and Buskirk 1982) and close to the limit of precision of measurement. The degree of aerobic fitness has been said to modify the relationship between altitude and the decrement in $\dot{V}O_2$ max (Buskirk et al. 1967; Buskirk 1969; Grover et al. 1967; Saltin 1967). Different studies have reported aerobic fitness to have opposite effects; two studies report the decrement to be greater in fit persons (Grover et al. 1967; Saltin 1967) while three report the decrement to be less in fit persons (Buskirk et al. 1967; Buskirk 1969; Hartley 1971). The purpose of this investigation was to determine the extent to which aerobic fitness influenced the size of the decrement in $\dot{V}O_2$ max at high altitude.

METHODS

Data collected from fifty-one male soldiers participating in several investigations were used in this study. Each subject had been fully informed as to the nature, requirements and potential risks of participation. All were

permanent residents of sea level (SL) who had not sojourned at high altitude (HA) for at least three months prior to their participation. The subjects' ages, heights, and weights were (mean \pm SE) 22 ± 1 yr, 177 ± 7 cm and 78 ± 2 kg, respectively.

$\dot{V}O_2$ max was determined at SL (50m) and again during the first eight days at HA (4,300). The $\dot{V}O_2$ max was measured at both SL and HA using a discontinuous cycling protocol (Kamon 1972) for 29 subjects, and a continuous cycling protocol (McArdle et al. 1973) for 22 subjects. For the discontinuous tests, subjects performed exercise bouts (approximately 4 min) of increasing intensity until an increase of 30W produced an increase in $\dot{V}O_2$ of less than $150 \text{ ml} \cdot \text{min}^{-1}$. For the continuous tests, exercise intensity was progressively increased at regular intervals until the subject could no longer continue. The highest $\dot{V}O_2$ measured during exercise was taken as maximum, and a plateau of $\dot{V}O_2$ with increased exercise intensity was obtained in the majority of these tests.

Open-circuit spirometry was used to measure oxygen uptake and carbon dioxide production. For the discontinuous tests, timed collections of expired air were made. Expired gas volumes were measured using a Tissot spirometer, and O_2 and CO_2 concentrations were measured using an oxygen fuel cell (Applied Electrochemistry S-3A) and infrared CO_2 (Beckman LB-2) gas analyzers, respectively. The gas analyzers were calibrated before each experiment using gases of known composition (mass spectrometer). For the continuous tests, respiratory exchange was determined every 30 seconds using a semi-automated system. Expired gas volumes were measured with a flow transducer (Hewlett-Packard) and a #3 pneumotach; O_2 and CO_2 concentrations were determined as before. Analog signals from the flow transducers and gas analyzers were digitized, and $\dot{V}O_2$, $\dot{V}CO_2$ (STPD) and \dot{V}_E (BTPS) were calculated using modified computational procedures of Sue et al. (1980).

Standard statistical procedures were used to analyze the data. The decrement in $\dot{V}O_2$ max at 4300 was calculated both as the absolute difference ($\text{ml} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$) between SL and HA, as well as the % of $\dot{V}O_2$ max at SL which was lost at HA. Body weight, $\dot{V}O_2$ max at SL and decrement in $\dot{V}O_2$ max at HA (both methods of computation) were tested for normality of distribution using the Chi-square goodness of fit test (Snedecor and Cochran 1967). Linear regression was used to determine the correlation between aerobic fitness and the decrement (both computations) in $\dot{V}O_2$ max at HA (Snedecor and Cochran 1967). The level of significance was $P < 0.05$.

RESULTS

The subjects' weights were normally distributed ($\chi^2 = 2.10$, $df = 2$, $0.25 < P < 0.50$). As shown in Figure 1, there was a slight tendency for subjects to weigh less than the group mean. The weights ranged from 55 to 109 kg with a mean \pm SE of 78 ± 2 kg.

FIGURES 1 AND 2 ABOUT HERE

Figure 2 shows the distribution of values of $\dot{V}O_2$ max at SL. $\dot{V}O_2$ max was normally distributed ($\chi^2 = 5.11$, $df = 4$, $0.25 < P < 0.50$) ranging from 36 to 60 $\text{ml} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$ with a mean \pm SE of 48 ± 1 $\text{ml} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$. The distribution of decrement in $\dot{V}O_2$ max at HA is shown in Figure 3. The absolute decrement (Figure 3A) in $\dot{V}O_2$ max at HA was normally distributed ($\chi^2 = 3.38$, $df = 3$, $0.25 < P < 0.50$) between 3 and 29 $\text{ml} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$ with a mean \pm SE of 13 ± 1 $\text{ml} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$. Similarly, the % of $\dot{V}O_2$ max at SL which was lost at

HA (Figure 3B) was normally distributed ($\chi^2 = 1.80$, $df = 5$, $0.75 < P < 0.90$) from 9 to 54% and averaged $27 \pm 1\%$.

FIGURE 3 ABOUT HERE

In Figure 4, the absolute decrement in $\dot{V}O_2$ max at HA is plotted as a function of the $\dot{V}O_2$ max at SL. The linear correlation coefficient (r) between these two variables was 0.56, which was statistically significant. However, only 31% of the variance in the absolute decrement was accounted for by the $\dot{V}O_2$ max at SL. The corresponding linear regression equation was $Y = 0.5X - 11.39$, where Y is the absolute decrement and X is the $\dot{V}O_2$ max at SL. Figure 5 shows the decrement expressed as % $\dot{V}O_2$ max at SL plotted as a function of the $\dot{V}O_2$ max at SL. The r for these two variables was 0.30. While this again was statistically significant, only 9% of the variance in the relative decrement was accounted for by the $\dot{V}O_2$ max at SL. The corresponding regression equation was $Y = 0.50X + 3.49$. Thus, the more aerobically fit men did lose a greater portion of their maximal aerobic power at HA.

FIGURE 4 and 5 ABOUT HERE

DISCUSSION

Maximal oxygen uptake is directly proportional to the maximum rate of systemic oxygen transport, which is the product of arterial oxygen content (C_aO_2) and maximum cardiac output (\dot{Q}_{max}). With acute (<24 hours) hypoxic exposure, \dot{Q}_{max} is unchanged from SL, but arterial oxygen saturation, thus C_aO_2 , is reduced resulting in a decrement in $\dot{V}O_2 \text{ max}$ (Horstman et al. 1980). After some days at HA, C_aO_2 returns towards normal (due to an increased hematocrit), but \dot{Q}_{max} becomes reduced (due to an decreased maximum stroke volume and heart rate) with the net effect that $\dot{V}O_2 \text{ max}$ remains depressed (Saltin et al. 1968).

Hartley (1971) calculated that, beginning at 1500 m above SL, $\dot{V}O_2 \text{ max}$ declines about 10% for each additional 1000 m ascended. There is, however, a good deal of variability in the decrement experienced, especially when moderate altitudes are considered. For example, Saltin (1967) reported that the decrement in $\dot{V}O_2 \text{ max}$ experienced by eight male athletes at 2250 m ranged from 9 to 22% of their $\dot{V}O_2 \text{ max}$ at SL, and in another study (Saltin et al. 1968) of four subjects at 4300 m, decrements were reported which ranged from 19 to 32%. In the present study, the decrement at 4300m averaged 27% of the $\dot{V}O_2 \text{ max}$ at SL, almost exactly the 28% which would be predicted based on the reports of Buskirk (1969) and Hartley (1971).

Although some investigators have reported that the level of aerobic fitness influences the magnitude of the decrement in $\dot{V}O_2 \text{ max}$ at HA, consensus is lacking. However, the previous investigations reporting a relationship between fitness and $\dot{V}O_2 \text{ max}$ at altitude were all based on studies of small numbers of subjects having a limited range of aerobic fitness. The present study indicates

that the degree of aerobic fitness, as indicated by $\dot{V}O_2 \text{ max}$, accounts for relatively little of the variability in the decrement in $\dot{V}O_2 \text{ max}$ at HA. This finding is based on results from a very large sample size having a wide range of individual aerobic fitness, body weight, and decrement in $\dot{V}O_2 \text{ max}$ at HA. The subject group was normally distributed with respect to these parameters. Irrespective of how the decrement is expressed ($\text{ml} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$, or % $\dot{V}O_2 \text{ max}$ at SL), most of the variability in the decrement at HA must be accounted for by factors other than physical fitness, per se.

A number of other such factors can be postulated. The physiological responses to extreme environmental heat (Drinkwater and Horvath 1979) and cold (Leblanc et al. 1978) are influenced to some degree by the age of the subject, so perhaps responses to hypoxia are also age-related. The present study cannot adequately address this issue since the age range of subjects was extremely narrow (18 to 31 years), but for this group there was no significant correlation between age and the decrement in $\dot{V}O_2 \text{ max}$ ($r = .03$). It could be suggested that the decrement in $\dot{V}O_2 \text{ max}$ is related to the length of time spent at HA, but the $\dot{V}O_2 \text{ max}$ of SL residents does not change during the first 15 days of HA residence (Young et al. 1982). The degree of arterial oxygen saturation during maximal exercise at altitude may vary between individuals. Dempsey et al. (1978) have reported that certain individuals at SL, notably elite track athletes, show significant hypoventilation and significant hypoxemia at or near their $\dot{V}O_2 \text{ max}$. These individuals possibly suffer a larger decrement in $\dot{V}O_2 \text{ max}$ at altitude as compared to normal individuals who do not experience arterial desaturation during maximal exercise. Individuals such as these were not included among the subjects of the present study, where only five subjects had $\dot{V}O_2 \text{ max}$ between 55 and $60 \text{ ml} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$ and none exceeded $60 \text{ ml} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$.

In conclusion, the results of this study indicate that the average decrement in maximal aerobic power of a group of SL residents acutely exposed to HA is reasonably well predicted by the relationship previously described by Buskirk (1969) and by Hartley (1971). There is a tendency for the more aerobically fit individual to incur a larger decrement in maximal aerobic power at high altitude. However, there is considerable interindividual variability in the observed decrement, whether expressed in absolute terms or relative to $\dot{V}O_2$ max at SL, and a relatively small amount of this variability is accounted for by the individual's level of aerobic fitness.

ACKNOWLEDGEMENT

Human subjects participated in this study after giving their free and informed voluntary consent. Investigators adhered to AR 70-25 and USAMRDC Regulation 70-25 on the use of volunteers in research. The views, opinions, and/or findings contained in this report are those of the authors and should not be construed as official Department of the Army position, policy, or decision unless so designated by other official documentation.

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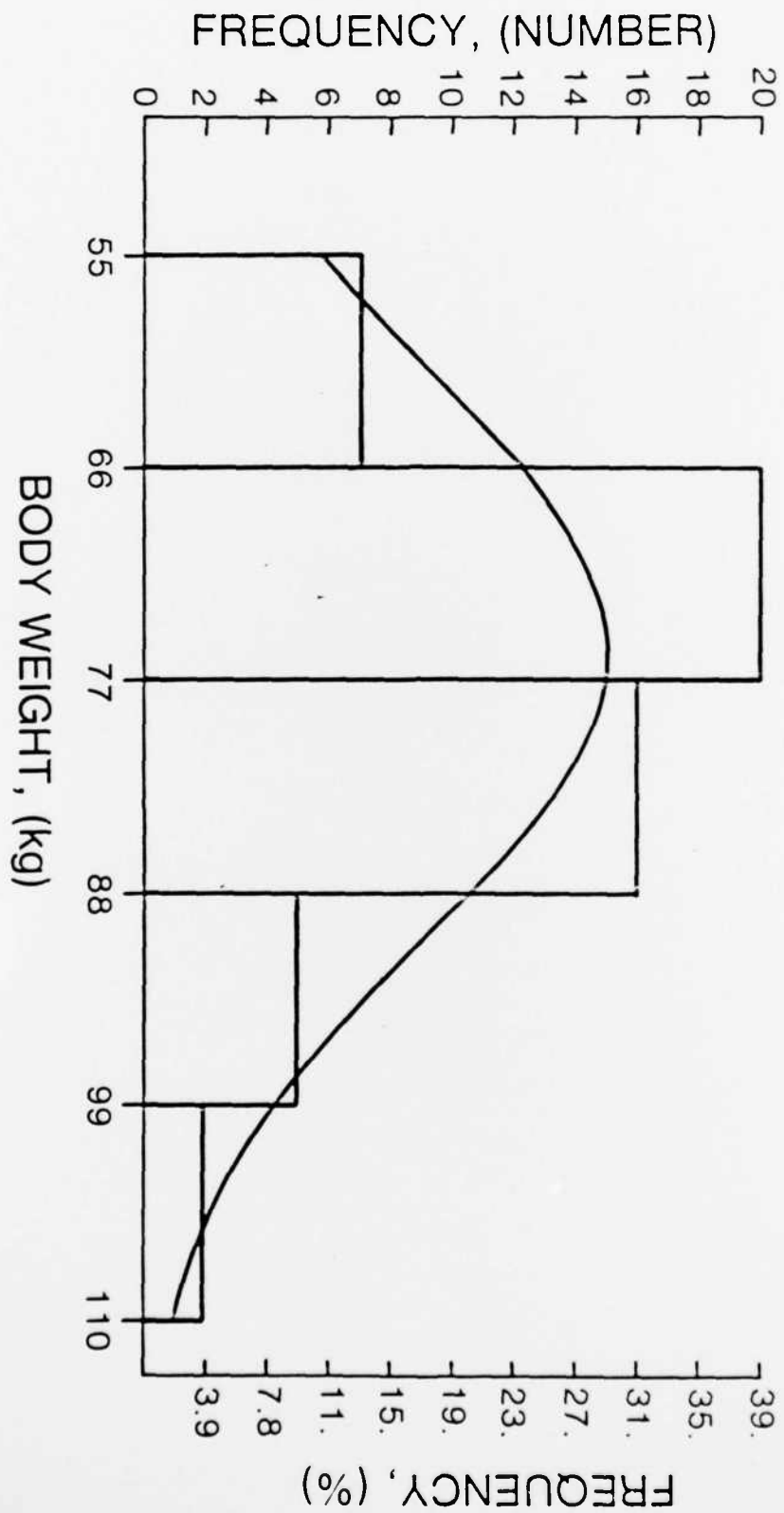
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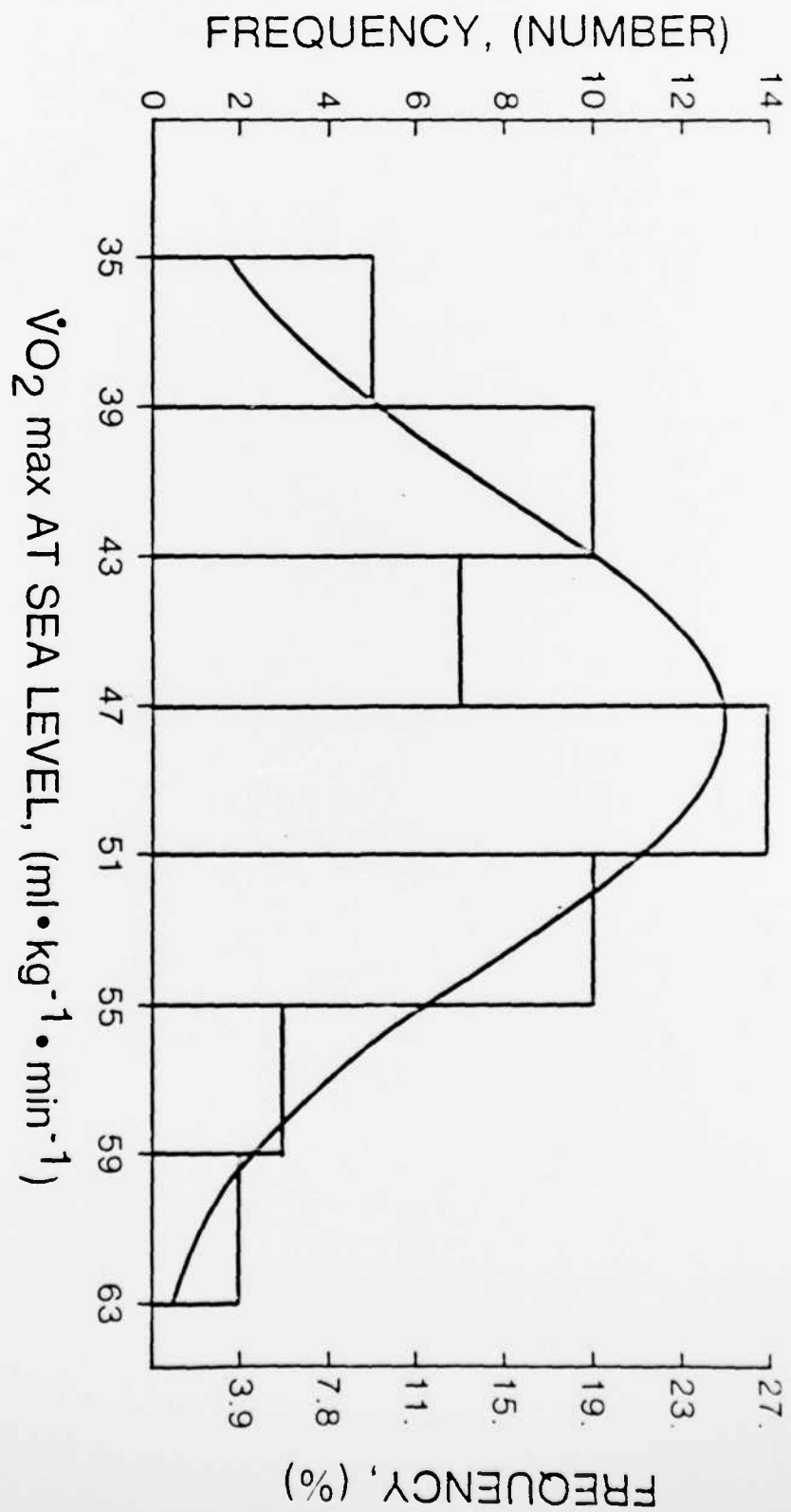
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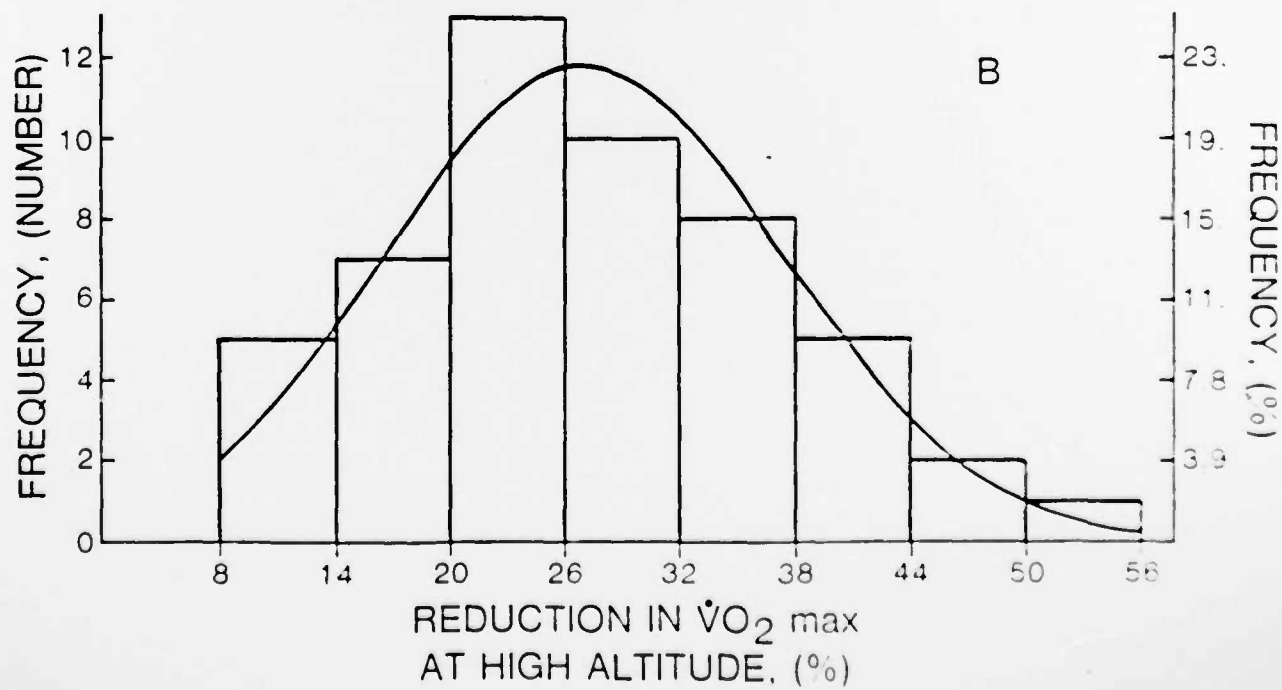
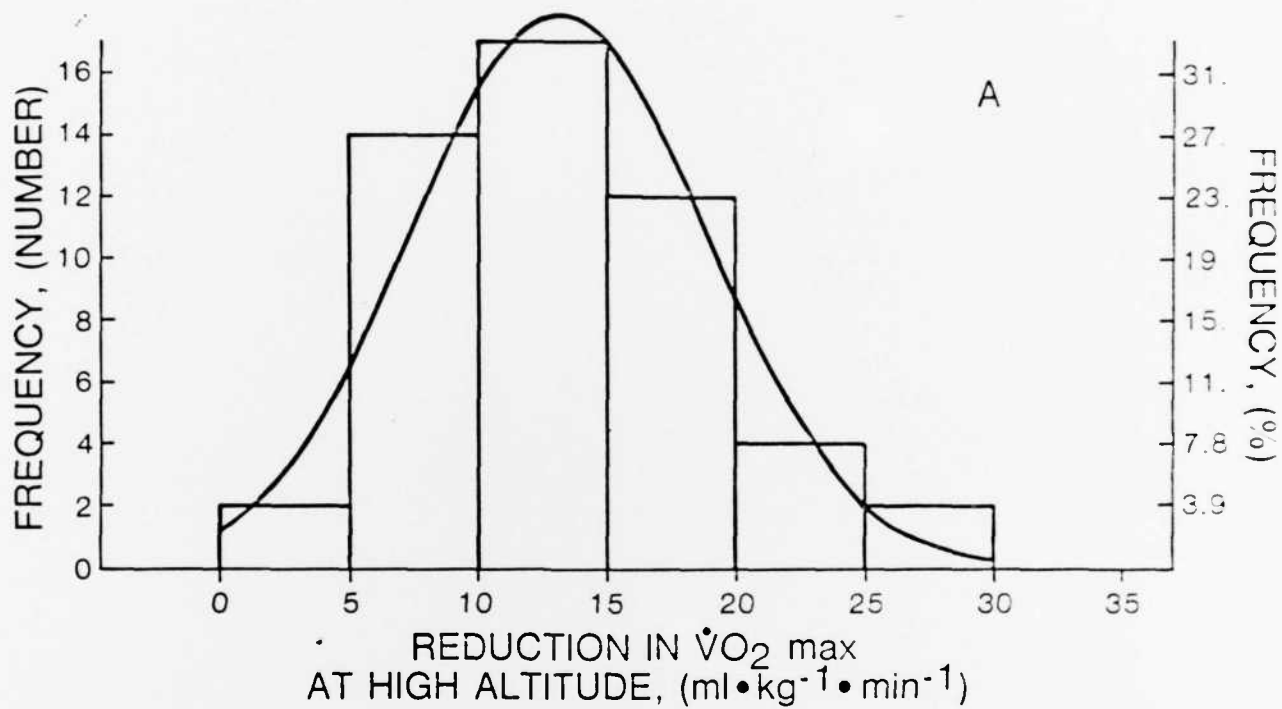
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FIGURE LEGENDS

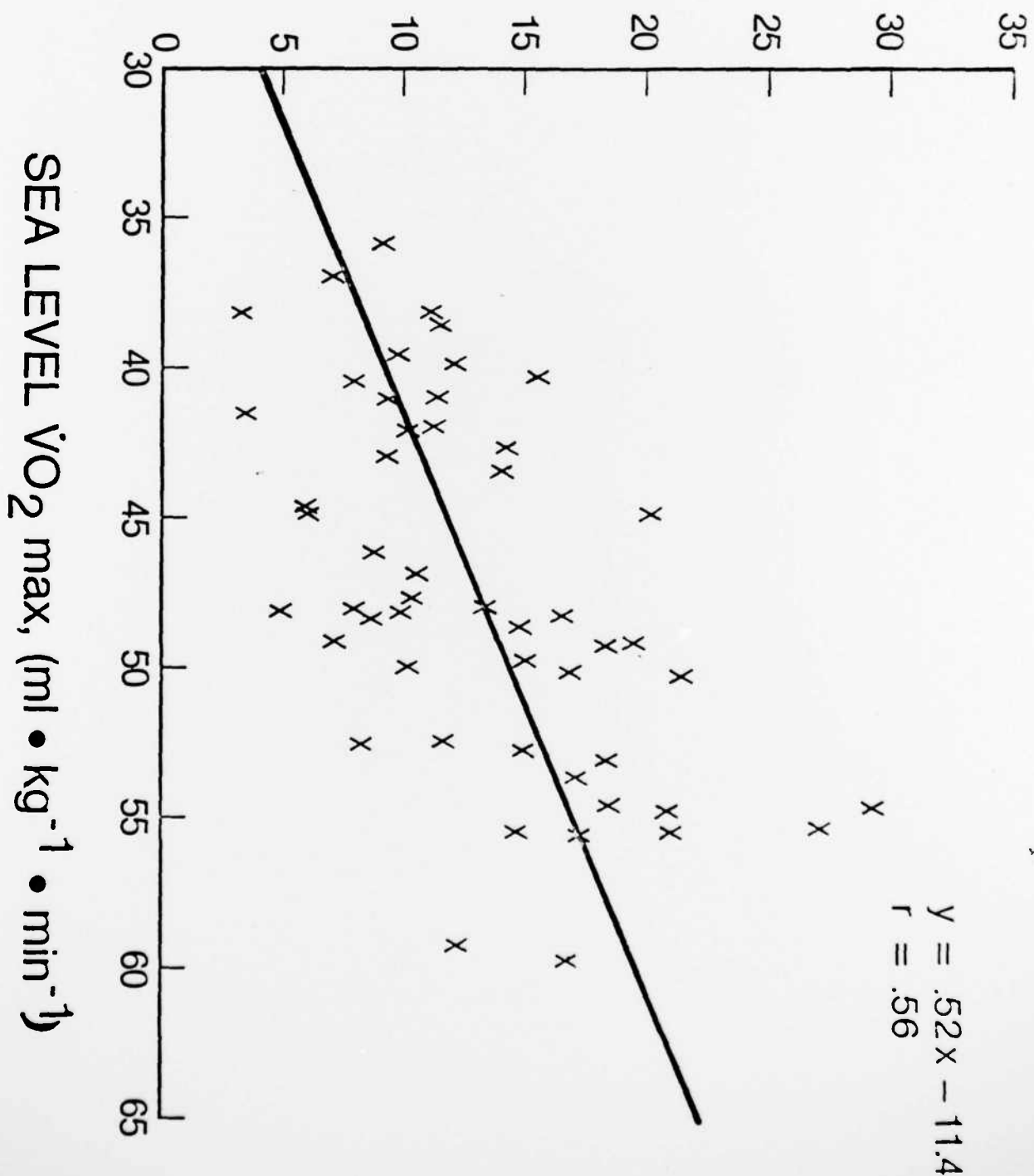
- Figure 1. Distribution of body weights for the test subject group (n=51).
- Figure 2. Distribution of maximal oxygen uptakes ($\dot{V}O_2 \text{ max}$), determined at sea level, for the test subject group (n=51).
- Figure 3. Distribution of the decrements in maximal oxygen uptake ($\dot{V}O_2 \text{ max}$) experienced by the subjects expressed as the absolute difference between sea level and high altitude (A) or expresses as the % difference (B).
- Figure 4. The absolute decrement in maximal oxygen uptake ($\dot{V}O_2 \text{ max}$) at high altitude plotted as a function of $\dot{V}O_2 \text{ max}$ at sea level. Solid line denotes regression line and individual values are denoted by X.
- Figure 5. The % decrement in maximal oxygen uptake ($\dot{V}O_2 \text{ max}$) at high altitude plotted as a function of $\dot{V}O_2 \text{ max}$ at sea level. Solid line denotes regression line and individual values are denoted by X.



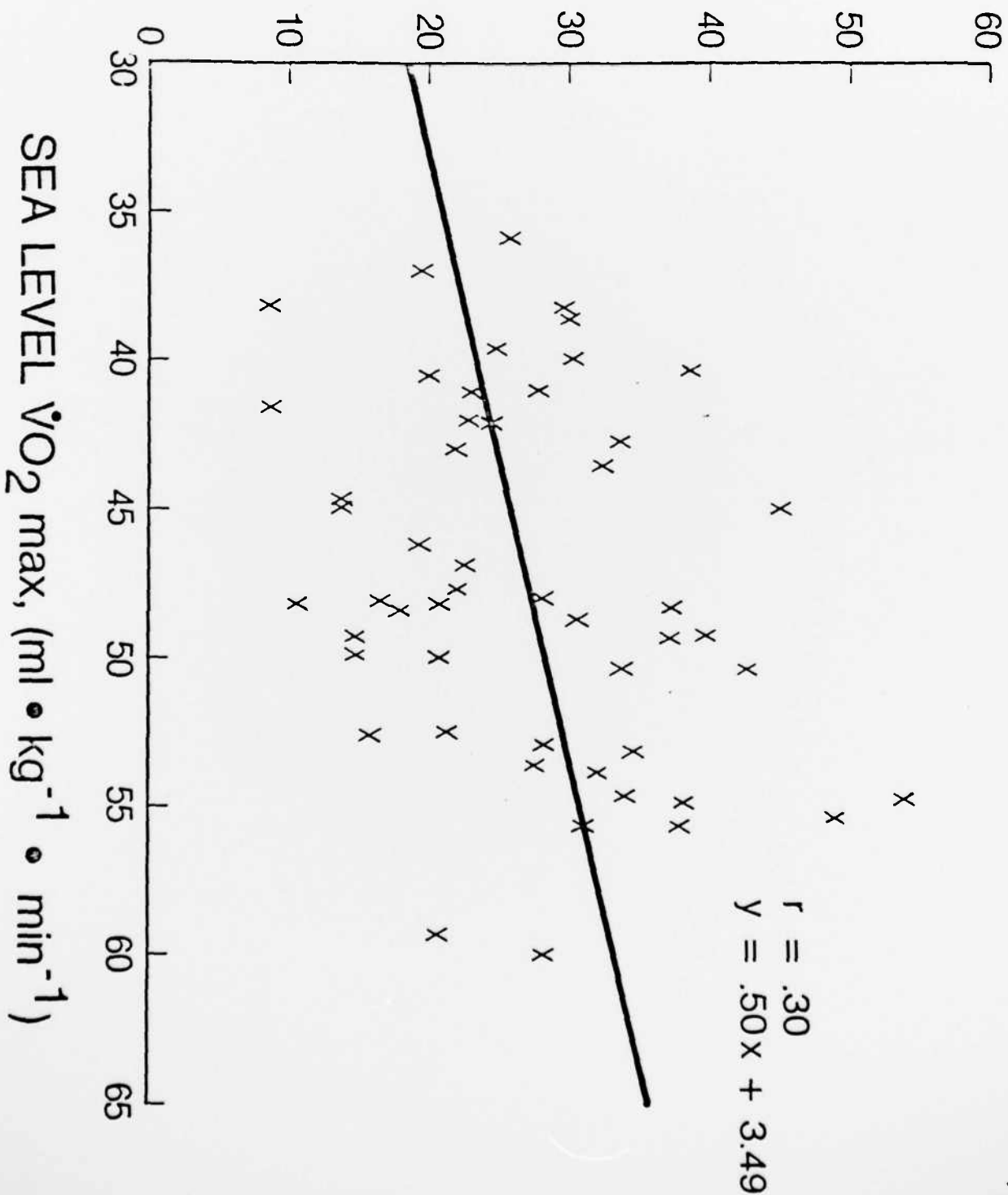




REDUCTION IN $\dot{V}O_2$ max
AT HIGH ALTITUDE, ($\text{ml} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$)



REDUCTION IN $\dot{V}O_2$ max AT HIGH ALTITUDE, (%)



DATE
FILME

FREQUENCY, (%)

39.
35.
31.
27.
23.
19.
15.
11.
7.8
3.9

FREQUENCY, (%)

